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B7-1S	0
B7.USPT.	6721
B7S	0
ANTIBOD$	0
ANTIBOD.USPT.	212
ANTIBODAY.USPT.	1
ANTIBODEES.USPT.	1
ANTIBODEIS.USPT.	1
ANTIBODES.USPT.	43
. . . . .	
TREAT$(TREATMENT/ULTRAFILTRATION).USPT.	pickup term
(('B7-1' OR 'B7') SAME ANTIBOD$ AND (INHIBIT$ OR TREAT$ OR SUPPRESS$ OR THERAP$) SAME (PSORIASIS)).USPT.	44

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- JPO Abstracts Database
- EPO Abstracts Database
- Derwent World Patents Index
- IBM Technical Disclosure Bulletins

Refine Search:

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('b7-1' or 'b7') same antibod$ and
(inhibit$ or treat$ or suppress$ or
therap$) same (psoriasis)
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Clear

Search History

Today's Date: 2/25/2001

DB Name	Query	Hit Count	Set Name
USPT	('b7-1' or 'b7') same antibod$ and (inhibit$ or treat$ or suppress$ or therap$) same (psoriasis)	44	L1

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     $0.01  TELNET
     $0.01  Estimated cost this search
     $0.33  Estimated total session cost   0.162 DialUnits

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  File   5:Biosis Previews(R)  1969-2003/Aug W1
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E7        8  AU=ANDERSON DARWIN W
E8        1  AU=ANDERSON DARYLL
E9        5  AU=ANDERSON DAVE
E10      82  AU=ANDERSON DAVID
E11      31  AU=ANDERSON DAVID A
E12       1  AU=ANDERSON DAVID ANDREW

          Enter P or PAGE for more
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              7  AU=ANDERSON DARRELL
              0  AU=ANDERSON DARRELL ?
              4  AU=ANDERSON DARRELL E
              7  AU=ANDERSON DARRELL R
              1  AU=ANDERSON DARWIN
              8  AU=ANDERSON DARWIN W
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? s s2 and (b7? or cd80 or cd86)
             27  S2
          22132  B7?
           9225  CD80
           7523  CD86
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S3 3 S2 AND (B7? OR CD80 OR CD86)
? t s3/3/all

3/3/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2003 BIOSIS. All rts. reserv.

13591346 BIOSIS NO.: 200200220167
Therapeutic activity of IDEC-114 (anti-**CD80**) and rituximab (Rituxan(R)) in B-cell lymphoma.
AUTHOR: Hariharan Kandasamy(a); **Anderson Darrell**(a); Leigh Bryan(a); Berquist Lisa G(a); Murphy Tracey(a); Leonard John E(a); Braslawsky Gary R(a); Hanna Nabil(a
AUTHOR ADDRESS: (a)IDEC Pharmaceuticals Corp., San Diego, CA**USA
JOURNAL: Blood 98 (11 Part 1):p608a November 16, 2001
MEDIUM: print
CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001
ISSN: 0006-4971
RECORD TYPE: Abstract
LANGUAGE: English

3/3/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2003 BIOSIS. All rts. reserv.

12995073 BIOSIS NO.: 200100202222
Human **B7**.1-specific primatized antibodies and transfectomas expressing said antibodies.
AUTHOR: **Anderson Darrell R**(a); Brams Peter; Hanna Nabil; Shestowsky William S; Heard Cheryl
AUTHOR ADDRESS: (a)Escondido, CA**USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1238 (1):pNo Pagination Sep. 5, 2000
MEDIUM: e-file
ISSN: 0098-1133
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

3/3/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2003 BIOSIS. All rts. reserv.

11995118 BIOSIS NO.: 199900275637
In vitro immunological and functional properties of primate/human chimeric monoclonal (PRIMATIZED(R)) antibodies to human **CD80**.
AUTHOR: Shestowsky William(a); Brams Peter(a); Pan Li-Zhen(a); Nguyen Mai-Lan(a); Chambers-Slater Karen(a); Franco Luis(a); Hanna Nabil(a); **Anderson Darrell**(a
AUTHOR ADDRESS: (a)IDEC Pharmaceuticals Corporation, 11011 Torreyana Road, San Diego, CA, 92121**USA
JOURNAL: FASEB Journal 13 (5 PART 2):pA953 March 15, 1999
CONFERENCE/MEETING: Annual Meeting of the Professional Research Scientists on Experimental Biology 99 Washington, D.C., USA April 17-21, 1999
SPONSOR: Federation of American Societies for Experimental Biology
ISSN: 0892-6638
RECORD TYPE: Citation
LANGUAGE: English
? s (b7? or cd80 or cd86) and (16C10 or 7C10 or 20C9 or 7B6)
22132 B7?
9225 CD80

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            7523  CD86
               1  16C10
              32  7C10
              16  20C9
              39  7B6
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...completed examining records
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? t s5/7/all

 5/7/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2003 BIOSIS. All rts. reserv.

12995073   BIOSIS NO.: 200100202222
Human B7.1-specific primatized antibodies and transfectomas
  expressing said antibodies.
AUTHOR: Anderson Darrell R(a); Brams Peter; Hanna Nabil; Shestowsky William
  S; Heard Cheryl
AUTHOR ADDRESS: (a)Escondido, CA**USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office
Patents  1238 (1):pNo Pagination Sep. 5, 2000
MEDIUM: e-file
ISSN: 0098-1133
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The present invention relates to the identification of macaque
  antibodies to human B7.1 and B7.2 by screening of phage
  display libraries or monkey heterohybridomas obtained using B lymphocytes
  from B7.1 and/or B7.2 immunized monkeys. More specifically,
  the invention provides four monkey monoclonal antibodies 7B6,
  16C10, 7C10 and 20C9 which inhibit the B7:CD28
  pathway and thereby function as effective immunosuppressants. The
  invention further provides the complete DNA and amino acid sequences of
  the light and heavy chain of three primatized antibodies derived from
  those monkey monoclonal antibodies which bind B7.1 and possibly
  B7.2, primatized 7C10, primatized 7B6 and primatized
  16C10. These primatized and monkey antibodies may be used as
  specific immunosuppressants, e.g., for the treatment of autoimmune
  diseases and to prevent organ transplant rejection.


 5/7/2     (Item 2 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2003 BIOSIS. All rts. reserv.

09114917   BIOSIS NO.: 199497123287
Induction of B cell costimulatory function by recombinant murine CD40
  ligand.
AUTHOR: Kennedy Mary K(a); Mohler Kendall M; Shanebeck Kurt D; Baum Peter R
  ; Picha Kathleen S; Otten-Evans Carol A; Janeway Charles A Jr; Grabstein
  Kenneth H
AUTHOR ADDRESS: (a)Dep. Immunobiol., Immunex Res. Dev. Corp., 51 University
  St., Seattle, WA 98101**USA
JOURNAL: European Journal of Immunology  24 (1):p116-123 1994
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: T cell-dependent regulation of B cell growth and differentiation
```

involves an interaction between CD40, a B cell surface molecule, and the CD40 ligand (CD40L) which is expressed on activated CD4+ T cells. In the current study, we show that recombinant membrane-bound murine CD40L induces B cells to express costimulatory function for the proliferation of CD4+ T cells. CD40L- or lipopolysaccharide (LPS)-activated, but not control-cultured B cells were strong costimulators of anti-CD3 or alloantigen-dependent T-cell responses. The molecular interactions responsible for the increased costimulatory functions were examined by analyzing the activated B cells for changes in the expression of two costimulatory molecules, **B7** and heat-stable antigen (HSA), as well as by the use of antagonists of **B7** and HSA (CTLA4.Fc and **20C9**, respectively). The expression of both **B7** and HSA was enhanced on B cells activated with LPS. As observed in previous studies, the costimulatory activity of the LPS-activated B cells was dependent on both **B7** and HSA and was completely inhibited in the presence of a combination of CTLA4.Fc and **20C9**. In contrast, activation of B cells with CD40L induced the expression of **B7** but did not enhance the expression of HSA. In addition the costimulatory activity of the CD40L-activated B cells was partially, but not completely, inhibited by the combination of CTLA4.Fc and **20C9**. These results demonstrate that CD40L regulates costimulatory function of B cells in part by inducing the expression of **B7** and suggest that CD40L-activated B cells express an additional costimulatory activity that is not associated with LPS-activated B cells.

5/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

08738923 BIOSIS NO.: 199395028274
Co-stimulation of murine CD4 T cell growth: Cooperation between **B7** and heat-stable antigen.
AUTHOR: Liu Yang(a); Jones Bryan; Brady William; Janeway Charles A Jr; Linley Peter S
AUTHOR ADDRESS: (a)Div. Immunol., Dep. Pathol., New York Univ. Med. Cent., 550 First Ave., New York, N.Y. 10016**USA
JOURNAL: European Journal of Immunology 22 (11):p2855-2860 1992
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The B cell activation antigen **B7**/BB1 has been shown to co-stimulate growth of human T cells by binding the T cell molecule CD28. In mice, the heaert-stable antigen (HSA) has also been shown to act as a co-stimulator for T cell growth. In this study, we have evaluated the contributions of **B7** and HSA to the co-stimulatory activity of antigen-presenting cells (APC). Mouse **B7** provides co-stimulatory activity for murine CD4 T cells in anti-CD3-induced proliferation. Human CTLA4Ig, a chimeric molecule comprising the extracellular region of CTLA-4 fused to an immunoglobulin C-gamma fragment, binds to murine **B7**. We, therefore, use human CTLA4Ig and the hamster anti-HSA monoclonal antibody **20C9** to analyze the relative contributions of **B7** and HSA to the co-stimulatory activity of murine spleen APC. Our data reveal that both murine **B7** and HSA are expressed by dendritic cells and by low-density spleen B cells. Either CTLA4Ig alone or anti-HSA alone inhibited CD4 T cell proliferation to anti-CD3 by gt 90%, while CTLA4Ig and anti-HSA together were far more efficient in inhibiting clonal expansion of CD4 T cells. These results demonstrate that functionally defined co-stimulation involves at least **B7** and HSA and suggest that signals delivered by **B7** and HSA synergize in promoting T cell growth.
? s (b7? or cd80 or cd86) and (psoriasis)

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          22132  B7?
           9225  CD80
           7523  CD86
          55704  PSORIASIS
      S6     166  (B7? OR CD80 OR CD86) AND (PSORIASIS)
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...examined 50 records  (50)
...examined 50 records  (100)
...examined 50 records  (150)
...completed examining records
      S7     108  RD S6 (unique items)
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7/7/16 (Item 16 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11985665 BIOSIS NO.: 199900266184
CTLA4Ig-mediated blockade of T-cell costimulation in patients with **psoriasis** vulgaris.
AUTHOR: Abrams Judith R(a); Lebwohl Mark G; Guzzo Cynthia A; Jegasothy Brian V; Goldfarb Michael T; Goffe Bernard S; Menter Alan; Lowe Nicholas J; Krueger Gerald; Brown Michael J; Weiner Russell S; Birkhofer Martin J; Warner Garvin L; Berry Karen K; Linsley Peter S; Krueger James G; Ochs Hans D; Kelley Susan L; Kang Sewon
AUTHOR ADDRESS: (a)Bristol-Myers Squibb Pharmaceutical Research Institute, 5 Research Parkway, Wallingford, CT, 064**USA
JOURNAL: Journal of Clinical Investigation 103 (9):p1243-1252 May, 1999
ISSN: 0021-9738
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Engagement of the **B7** family of molecules on antigen-presenting cells with their T cell-associated ligands, CD28 and CD152 (cytotoxic T lymphocyte-associated antigen-4 (CTLA-4)), provides a pivotal costimulatory signal in T-cell activation. We investigated the role of the CD28/CD152 pathway in **psoriasis** in a 26-week, phase I, open-label dose-escalation study. The importance of this pathway in the generation of humoral immune responses to T cell-dependent neoantigens, bacteriophage phiX174 and keyhole limpet hemocyanin, was also evaluated. Forty-three patients with stable **psoriasis** vulgaris received 4 infusions of the soluble chimeric protein CTLA4Ig (BMS-188667). Forty-six percent of all study patients achieved a 50% or greater sustained improvement in clinical disease activity, with progressively greater effects observed in the highest-dosing cohorts. Improvement in these patients was associated with quantitative reduction in epidermal hyperplasia, which correlated with quantitative reduction in skin-infiltrating T cells. No markedly increased rate of intralesional T-cell apoptosis was identified, suggesting that the decreased number of lesional T cells was probably likely attributable to an inhibition of T-cell proliferation, T-cell recruitment, and/or apoptosis of antigen-specific T cells at extralesional sites. Altered antibody responses to T cell-dependent neoantigens were observed, but immunologic tolerance to these antigens was not demonstrated. This study illustrates the importance of the CD28/CD152 pathway in the pathogenesis of **psoriasis** and suggests a potential therapeutic use for this novel immunomodulatory approach in an array of T cell-mediated diseases.

7/7/13 (Item 13 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12465656 BIOSIS NO.: 200000219158
Results of a single-dose, dose-escalating trial of an anti-**B7**.1 monoclonal antibody (IDEC-114) in patients with **psoriasis**.
AUTHOR: Gottlieb A(a); Abdulghani A; Totoritis M; Lizambri R; Shuey S; Romano P; Oh C; Chaudhari U; Lebwohl M
AUTHOR ADDRESS: (a)UMDNJ-RWJMS, New Brunswick, NJ**USA
JOURNAL: Journal of Investigative Dermatology 114 (4):p840 April, 2000
CONFERENCE/MEETING: 61st Annual Meeting of the Society for Investigative Dermatology. Chicago, Illinois, USA May 10-14, 2000
ISSN: 0022-202X
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English

12552653 BIOSIS NO.: 200000306155
The role of co-stimulation in airway inflammation.
AUTHOR: Djukanovic R
AUTHOR ADDRESS: (a)University of Medicine, Southampton General Hospital, Tremona Road, Level D, Centre Block, Southampton, SO16 6YD**UK
JOURNAL: Clinical and Experimental Allergy 30 (Supplement 1):p46-50 June, 2000
MEDIUM: print
ISSN: 0954-7894
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: There is considerable evidence to support an important role for co-stimulatory molecules in regulating the proliferation and activation of T cells in the immune response. Of particular relevance is the interaction between CD28 on T cells and **B7** expressed on the surface of antigen presenting cells (APCs). CTLA-4, another molecule present on activated T cells may downregulate T cell activity, but its role remains uncertain. CTLA4-Ig, a fusion protein consisting of the extracellular domain of CTLA4 and the Fc portion of human immunoglobulin G1 (IgG1), has been useful for studying the role of CD28/**B7** interactions in immune responses. A number of studies have shown that CTLA4-Ig can switch off T cell activation. In an ovalbumin sensitive murine model of asthma, CTLA4-Ig treatment suppressed the response to inhaled allergen (increased airway hyperresponsiveness (AHR), IgE production, recruitment of eosinophils into the lungs, production of IL-4, IL-5, and IL-10 and increased IFNgamma production from CD3-TCR-activated T cells). Anti **B7**-2 treatment has similar effects suggesting that interaction of **B7**-2 with CD28 is important in the development of a Th-2 type inflammatory response in mice. Recent observations have been of relevance to human allergic disease. In vitro studies have shown that CTLA4-Ig or anti-**B7**-2 antibody can inhibit allergen-induced proliferation and cytokine production by peripheral blood mononuclear cells from atopic subjects. The role of co-stimulation has been studied in a human bronchial explant model of asthma. CTLA4-Ig fusion protein effectively blocked allergen-induced production of IL-5 and IL-13 in bronchial explants from atopic asthmatics. These studies confirm the requirement for interaction between co-stimulatory molecules in cytokine production and allergic inflammation, and point to the CD28-**B7** pathway as being important to the allergen-induced inflammation in asthma. Studies of organ transplantation in primates suggest that CTLA4-Ig is extremely effective in preventing organ rejection. While phase 1 clinical trials have shown CTLA-4-Ig treatment of patients with **psoriasis** vulgaris to be well tolerated and to result in clinical improvement, its role in asthma management merits further investigation.

12666545 BIOSIS NO.: 200000420047
Blockade of T lymphocyte costimulation with cytotoxic T lymphocyte-associated antigen 4-immunoglobulin (CTLA4Ig) reverses the cellular pathology of psoriatic plaques, including the activation of keratinocytes, dendritic cells, and endothelial cells.
AUTHOR: Abrams Judith R(a); Kelley Susan L; Hayes Elizabeth; Kikuchi Toyoko ; Brown Michael J; Kang Sewon; Lebwohl Mark G; Guzzo Cynthia A; Jegasothy Brian V; Linsley Peter S; Krueger James G
AUTHOR ADDRESS: (a)Novartis Pharmaceuticals Corporation, 59 Route 10, Bldg. 122, Rm. S320, East Hanover, NJ, 07936-1080**USA
JOURNAL: Journal of Experimental Medicine 192 (5):p681-693 September 4, 2000
MEDIUM: print
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Efficient T cell activation is dependent on the intimate contact between antigen-presenting cells (APCs) and T cells. The engagement of the **B7** family of molecules on APCs with CD28 and CD152 (cytotoxic T lymphocyte-associated antigen 4 (CTLA-4)) receptors on T cells delivers costimulatory signal(s) important in T cell activation. We investigated the dependence of pathologic cellular activation in psoriatic plaques on **B7**-mediated T cell costimulation. Patients with **psoriasis** vulgaris received four intravenous infusions of the soluble chimeric protein CTLA4Ig (BMS-188667) in a 26-wk, phase I, open label dose escalation study. Clinical improvement was associated with reduced cellular activation of lesional T cells, keratinocytes, dendritic cells (DCs), and vascular endothelium. Expression of CD40, CD54, and major histocompatibility complex (MHC) class II HLA-DR antigens by lesional keratinocytes was markedly reduced in serial biopsy specimens. Concurrent reductions in **B7**-1 (**CD80**), **B7**-2 (**CD86**), CD40, MHC class II, CD83, DC-lysosomal-associated membrane glycoprotein (DC-LAMP), and CD11c expression were detected on lesional DCs, which also decreased in number within lesional biopsies. Skin explant experiments suggested that these alterations in activated or mature DCs were not the result of direct toxicity of CTLA4Ig for DCs. Decreased lesional vascular ectasia and tortuosity were also observed and were accompanied by reduced presence of E-selectin, P-selectin, and CD54 on vascular endothelium. This study highlights the critical and proximal role of T cell activation through the **B7**-CD28/CD152 costimulatory pathway in maintaining the pathology of **psoriasis**, including the newly recognized accumulation of mature DCs in the epidermis.

7/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13267288 BIOSIS NO.: 200100474437
CD80/86 antisense oligonucleotide therapy improves **psoriasis**:
Study in the SCID mouse model.
AUTHOR: Raychaudhuri S(a); Bennett C; Karras J; Mehta R; Chatterjee A(a);
Farber E(a)
AUTHOR ADDRESS: (a)Psoriasis Research Institute, Palo Alto, CA**USA
JOURNAL: Journal of Investigative Dermatology 117 (2):p443 August, 2001
MEDIUM: print
CONFERENCE/MEETING: 62nd Annual Meeting of the Society for Investigative
Dermatology Washington, DC, USA May 09-12, 2001
ISSN: 0022-202X
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English

13877222 BIOSIS NO.: 200200506043
Differentiation of monocyte-derived dendritic cells in patients with **psoriasis**.
AUTHOR: Zhu Kejian(a); Zhou Weifang(a); Lao Limin(a); Zheng Min(a)
AUTHOR ADDRESS: (a)Department of Dermatology, Second Affiliated Hospital, Zhejiang University School of Medicine, Hangzhou, 310009**China
JOURNAL: Zhonghua Pifuke Zazhi 35 (2):p88-90 April, 2002
MEDIUM: print
ISSN: 0412-4030
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: Chinese; Non-English

ABSTRACT: Objective To investigate the differentiation of dendritic cells from monocytes of the peripheral blood in patients with **psoriasis**. Methods Flow cytometry was used to analyze the phenotype of monocyte-derived dendritic cells (MoDC). The capacity of MoDC to stimulate the proliferation of T lymphocytes was evaluated by allogeneic mixed lymphocyte reaction. Results Monocytes in the peripheral blood of patients with **psoriasis** could differentiate into dendritic cells. Expression of CD40, **CD80**, **CD86** and HLA-DR by MoDC was significantly increased in patients with **psoriasis** compared with that in normal controls ($P < 0.01$). The capacity of MoDC to stimulate T lymphocytes was enhanced significantly in patients' group as well ($P < 0.01$). Conclusions The differentiation of monocytes into dendritic cells is upregulated in **psoriasis**, and these cells have potent antigen presenting capacities.